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Efficacy of interposed abdominal compression-cardiopulmonary resuscitation (CPR), active compression and decompression-CPR, and Lifestick CPR: Basic physiology in a spreadsheet model

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ABSTRACT

This study was undertaken to understand and predict results of experimental cardiopulmonary resuscitation (CPR) techniques involving compression and decompression of either the chest or the abdomen. Simple mathematical models of the adult human circulation were used. Assumptions of the models are limited to normal human anatomy and physiology, the definition of compliance (volume change/pressure change), and Ohm's law (flow = pressure / resistance). Interposed abdominal compression-CPR, active compression and decompression of the chest, and Lifestick CPR, which combines interposed abdominal compression and active compression and decompression, produce, respectively, 1.9-, 1.2-, and 2.4- fold greater blood flow than standard CPR and systemic perfusion pressures of 45, 30, and 58 mm Hg, respectively. These positive effects are explained by improved pump priming and are consequences of fundamental principles of cardiovascular physiology.

KEY WORDS: active compression and decompression-cardiopulmonary resuscitation; ACD-CPR; blood flow; cardiopulmonary resuscitation; computers; heart arrest; interposed abdominal compression-cardiopulmonary resuscitation; IAC-CPR; Lifestick; mechanics

Crit Care Med 2000 Vol. 28, No. 11 (Suppl.) pp. N119-N202

Presented, in part, at the Wolf Creek V Conference, September 1999, Rancho Mirage, CA.

INTRODUCTION

Both interposed abdominal compression and active chest decompression have been proposed as effective means of augmenting perfusion during external cardiopulmonary resuscitation (CPR) (1– 3), (4–6). During interposed abdominal compression (IAC)-CPR, positive pressure is applied to the abdomen in counterpoint to the rhythm of chest compression, so that the abdomen is being compressed when chest pressure is relaxed.

During active compression-decompression (ACD)-CPR, positive and negative pressures are applied alternately to the chest by means of a “plunger” that forms a seal with the anterior chest wall. Both methods improve hemodynamics in animal studies (7, 8). Both improve CO₂ excretion as a measure of effective systemic perfusion in human resuscitations (4, 9, 10).

Three randomized clinical trials of IAC-CPR compared to standard CPR (2, 9, 11) have found statistically significant benefit, and one early trial found no difference (12). Four randomized clinical trials of ACD-CPR have found improved outcome (5, 6, 13, 14), and four other trials have found no difference (15–18). Recently, Lifestick CPR (19) has become the subject of active research, utilizing a two-handled device that is able simultaneously to apply IAC- and ACD-CPR, by alternately compressing and decompressing the chest and the abdomen through adhesive pads. These adjunctive maneuvers offer the promise of improved CPR, but are they “for real,” and what is their ultimate theoretical potential?

One approach to these questions is to determine whether the observed benefits of IAC and ACD are related to fundamental principles of cardiovascular physiology. If so, they are likely to work in most patients, despite clinical vagaries. To pursue the fundamental hemodynamic issues, the author created a simple mathematical model to integrate knowledge from the literature and to provide an independent comparison of the various CPR adjuncts in the same test system. Such a model is free from the many confounding factors present in laboratory studies and in clinical trials. These include varying patient populations, down time, drug therapy, central venous pressure, peripheral vascular resistance, underlying disease, chest configuration, and body size, as well as varying rescuer size, skill, strength, consistency, prior training, and bias. Mathematical models also allow exact control of the dominant hemodynamic mechanism of CPR (thoracic pump in large subjects vs. cardiac pump in small subjects) (20). This approach facilitates quantitative comparison of various resuscitation techniques in exactly the same test system.

MATERIALS AND METHODS

To capture the essence of CPR hemodynamics, one may solve the family of differential equations describing pressures and flows in a simplified circulatory system, the technical details and mathematics of which have been published elsewhere (21). In this physiologic model, the human circulation is represented by seven compliant chambers, connected by resistances through which blood may flow. The compliances correspond to the thoracic aorta, abdominal aorta, superior vena cava and right heart, abdominal and lower extremity veins, carotid arteries, and jugular veins. In addition, the chest compartment contains a pump representing the pulmonary vascular and left heart compliances. This pump may be configured to function either as a heart-like cardiac pump, in which applied pressure squeezes blood from the heart itself through the aortic valve, or as a global thoracic pressure pump, in which applied pressure squeezes blood from the pulmonary vascular bed, through the left heart, and into the periphery (22–24). Values for physiologic variables describing a textbook normal “70-kg man” (25) are used to specify compliances and resistances in the model (21). The distribution of vascular conductances (1/resistances) into cranial, thoracic, and caudal components reflects textbook distributions of cardiac output to various body regions.

Using a standard spreadsheet program, such as Microsoft Excel, it is easy to solve the equations for the circulatory model to obtain pressures throughout the system as a function of time. (The simple spreadsheet model presented here can be duplicated by anyone with a personal computer and ordinary business software. A version is available on the Internet [URL: <http://www.vet.purdue.edu/iaccpr>] or via e-mail from the author.) Although any arbitrary function or waveform can be used to represent the imposed chest and abdominal pressures in external CPR, the results described here were generated using half-sinusoidal functions to represent chest or abdominal compression or decompression.

To explore the influence of the thoracic pump vs. the cardiac pump mechanisms that can impel blood during cardiac arrest and chest compression, a factor, $0 \leq \text{Tpfactor} \leq 1$, is used. A pressure equal to the product of internal chest pressure and Tpfactor is applied to the thoracic aorta and superior vena cava to create a continuum of hybrid pump mechanisms ranging from pure cardiac pump (Tpfactor = 0) to pure thoracic pump (Tpfactor = 1). When Tpfactor = 1, all intrathoracic structures, including the great veins and thoracic aorta, experience a uniform “global” intrathoracic pressure rise, as originally conceived by Rudikoff et al (26). When Tpfactor = 0, only the pump compliance is pressurized, as if the heart alone, and not the great vessels, were compressed between the sternum and the spine, as originally conceived by Kouwenhoven et al (27). Intermediate values of the thoracic pump factor allow models approximating the current understanding (24, 28, 29), in which for small animals and children blood is impelled in external CPR predominantly by the cardiac pump mechanism (for example, Tpfactor = 0.25), whereas, in larger animals and adult humans, blood is impelled predominantly by the thoracic pump mechanism (for example, Tpfactor = 0.75).

The spreadsheet code was validated by establishing a model of the normal adult circulation using Tpfactor = 0. This model had an aortic blood pressure of 120/82 mm Hg and a cardiac output of 4.9 L/min for a heart rate of 80 beats/min, closely approximating the textbook normal values of 120/80 mm Hg and 5.0 L/min.

RESULTS

Figures 1–3 illustrate five-channel pressure records after 20 cycles of standard CPR, IAC-CPR, or ACD-CPR. The peak positive abdominal pressure for IAC-CPR is 110 mm Hg, and the maximal negative intrathoracic pressure for ACD-CPR is –20 mm Hg, approximating published values for the two techniques (1, 2, 30–33). In this model, the thoracic pump factor is 0.75 to simulate an adult patient, in whom the thoracic pump mechanism is dominant, but there is some degree of selective cardiac compression (24). For reference, Figure 1 illustrates pressures generated by standard CPR.

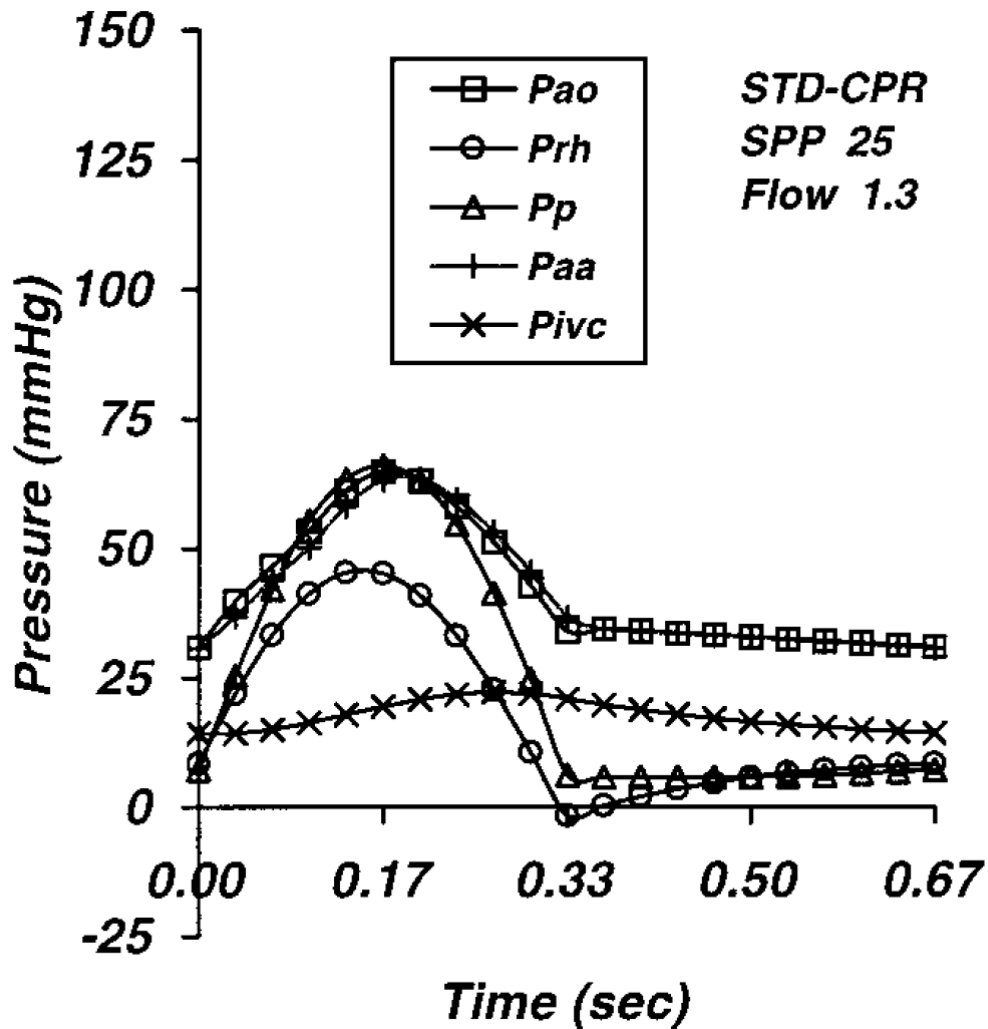


Figure 1. Pressures generated by standard cardiopulmonary resuscitation (*STD-CPR*) in a mathematical model. Steady state pressures after 20 compression cycles are shown. P_{ao} , thoracic aortic pressure; P_{rh} , right heart pressure; P_p , thoracic pump pressure; P_{aa} , abdominal aortic pressure; P_{ivc} , inferior vena cava pressure. Thoracic pump factor is 0.75. Peak applied pressures are: chest compression, +60; chest decompression, -20; abdominal compression, +110; abdominal decompression, -30. Compression rate is 90/min. *Flow*, forward flow in L/min; *SPP*, mean systemic perfusion pressure in mm Hg.

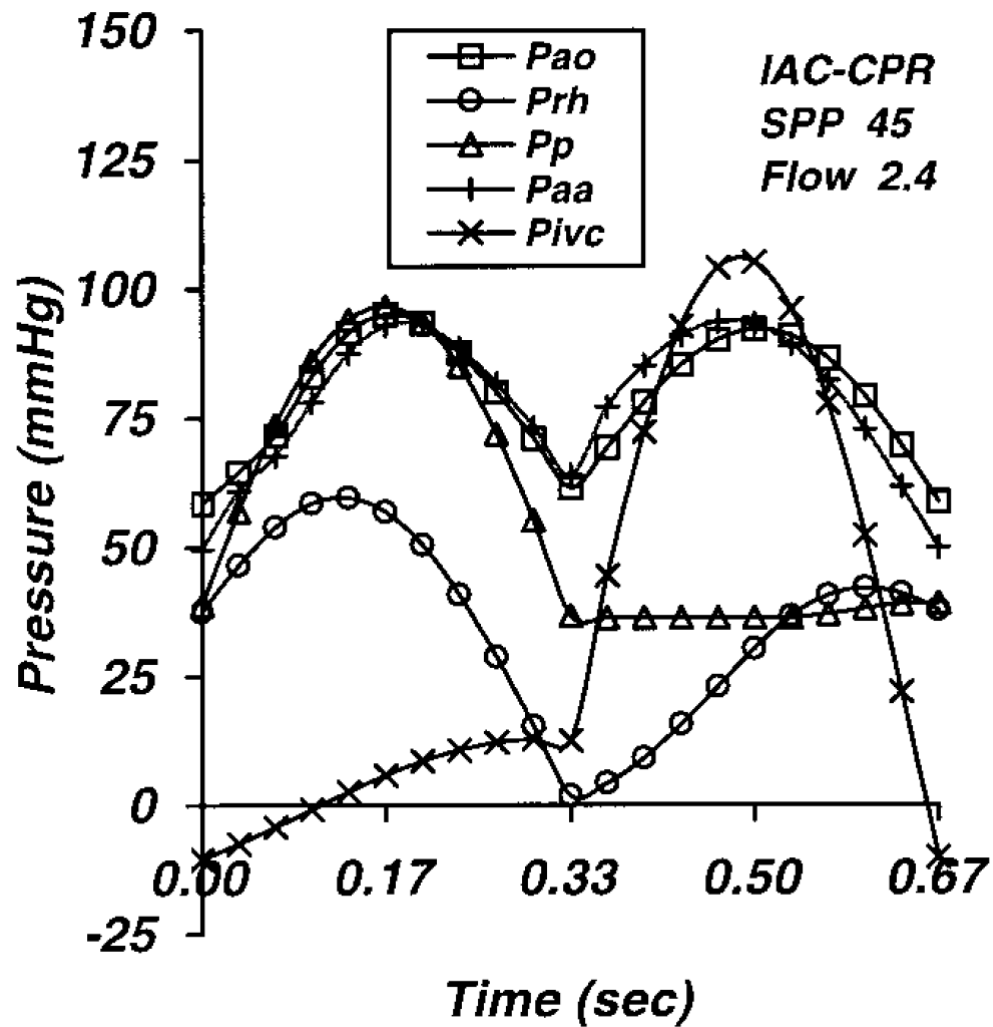


Figure 2. Pressures generated by interposed abdominal compression-cardiopulmonary resuscitation (*IAC-CPR*) in a mathematical model. Details are similar to Figure 1. P_{ao} , thoracic aortic pressure; P_{rh} , right heart pressure; P_p , thoracic pump pressure; P_{aa} , abdominal aortic pressure; P_{ivc} , inferior vena cava pressure; $Flow$, forward flow in L/min; SPP , mean systemic perfusion pressure in mm Hg.

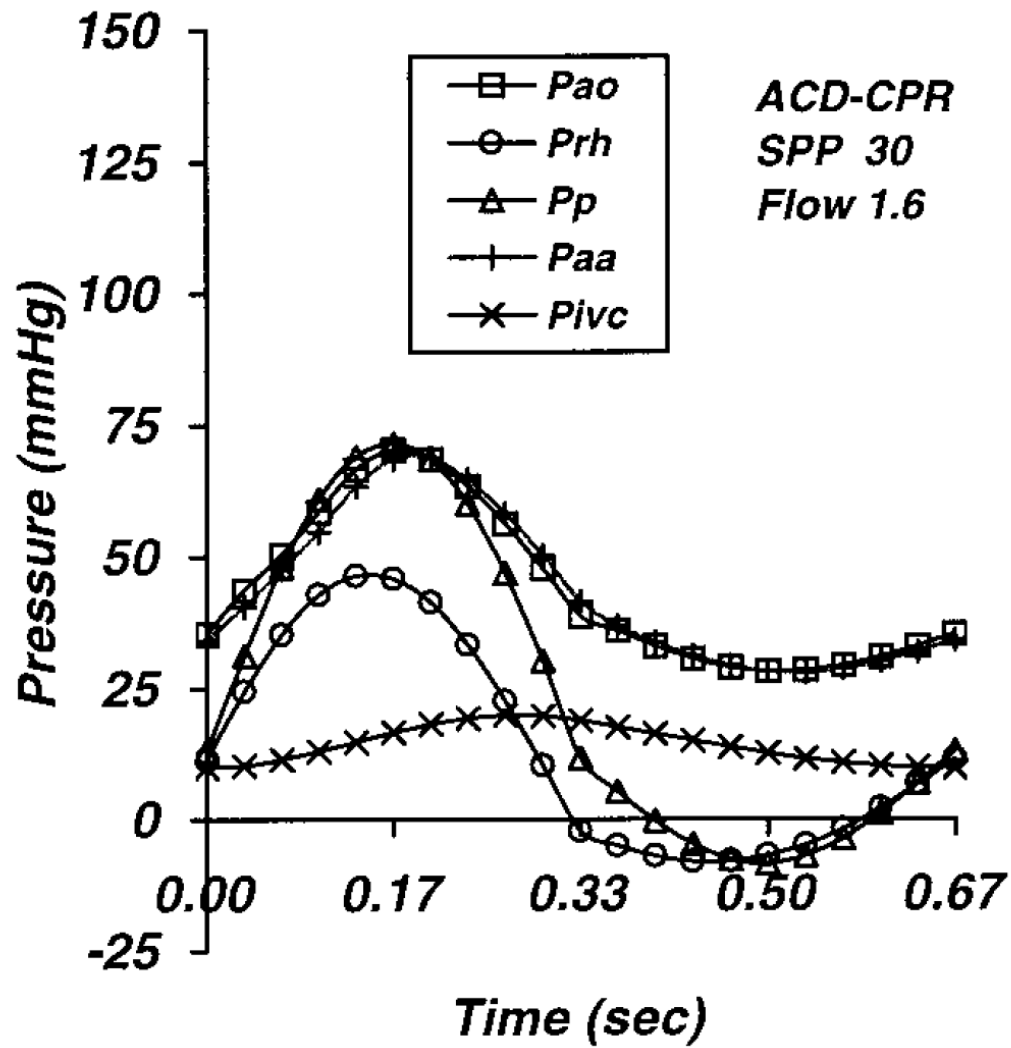


Figure 3. Pressures generated by active compression and decompression-cardiopulmonary resuscitation (*ACD-CPR*) in a mathematical model. Details are similar to Figure 1. P_{ao} , thoracic aortic pressure; P_{rh} , right heart pressure; P_p , thoracic pump pressure; P_{aa} , abdominal aortic pressure; P_{ivc} , inferior vena cava pressure; $Flow$, forward flow in L/min; SPP , mean systemic perfusion pressure in mm Hg.

IAC-CPR. Comparison of the pressure waveforms in Figures 1 and 2 elucidates the mechanism of +110 mm Hg interposed abdominal compression CPR. The abdominal venous pressure pulse induces increased right heart filling pressure during IAC and consequent faster pump emptying during chest compression, compared with standard CPR. Faster pump filling is caused by larger pressure gradients across the input valve from 0.54 to 0.67 sec of the cycle. Faster pump emptying is caused by the Starling characteristic of the pump associated with greater filling, and in turn, larger pressure gradients across the aortic valve during ejection. With the addition of IAC cardiac output increases from 1.3 to 2.4 L/min, and mean systemic perfusion pressure (SPP) increases from 25 to 45 mm Hg, compared to standard CPR. The abdominal aortic pressure waveform (crosses) leads the thoracic aortic pressure waveform (squares), during the onset and peak of IAC, indicating retrograde flow in the aorta.

ACD-CPR. Figure 3 shows steady-state pressure waveforms for -20 mm Hg ACD-CPR. Reduced pump pressure (pulmonary vascular pressure) during diastole promotes faster pump filling from 0.45 to 0.60 sec into the cycle. Cardiac output is increased from 1.3 to 1.6 L/min, and mean systemic perfusion pressure is increased from 25 to 30 mm Hg, compared with standard CPR. The ACD induced decrease in central venous pressure offsets the ACD-induced decrease in thoracic aortic pressure so that augmented perfusion pressure is maintained. Effects on systemic perfusion pressure of IAC and ACD are similar to those reported in experimental animals and in human patients (1, 30, 34–37).

Compared with standard CPR, 110 mm Hg IAC produced an 85% increase in total flow. In the same model, -20 mm Hg ACD produced a 23% increase in total flow. The present results in an independent mathematical model confirm that the positive findings in animal studies and most clinical studies are valid and are based on the fundamental anatomy and physiology of the circulatory system.

Four-Phase Lifestick CPR. Lifestick CPR is a recently developed technique for potentially combining the effects of IAC and ACD. The sticky, self-adhesive compression pads of the Lifestick permit active compression and decompression of both the chest and the abdomen. Accurate simulation of Lifestick CPR is difficult because actual values of negative intra-abdominal pressure have not yet been reported. If one estimates maximal decompression phase pressure in the abdomen to be -30 mm Hg, the results in Figure 4 are obtained. With this possible four-phase technique, mean systemic perfusion pressure is 58 mm Hg. Total forward flow is 3.1 L/min—2.5 times that of standard CPR. Examination of the pressure waveforms in Figure 4 reveals that in four-phase CPR, negative inferior vena cava pressure draws blood out of the chest from 0 to 0.3 sec into the cycle, widening the systemic perfusion pressure. Positive inferior vena cava pressure from 0.33 to 0.67 sec promotes excellent pump filling.

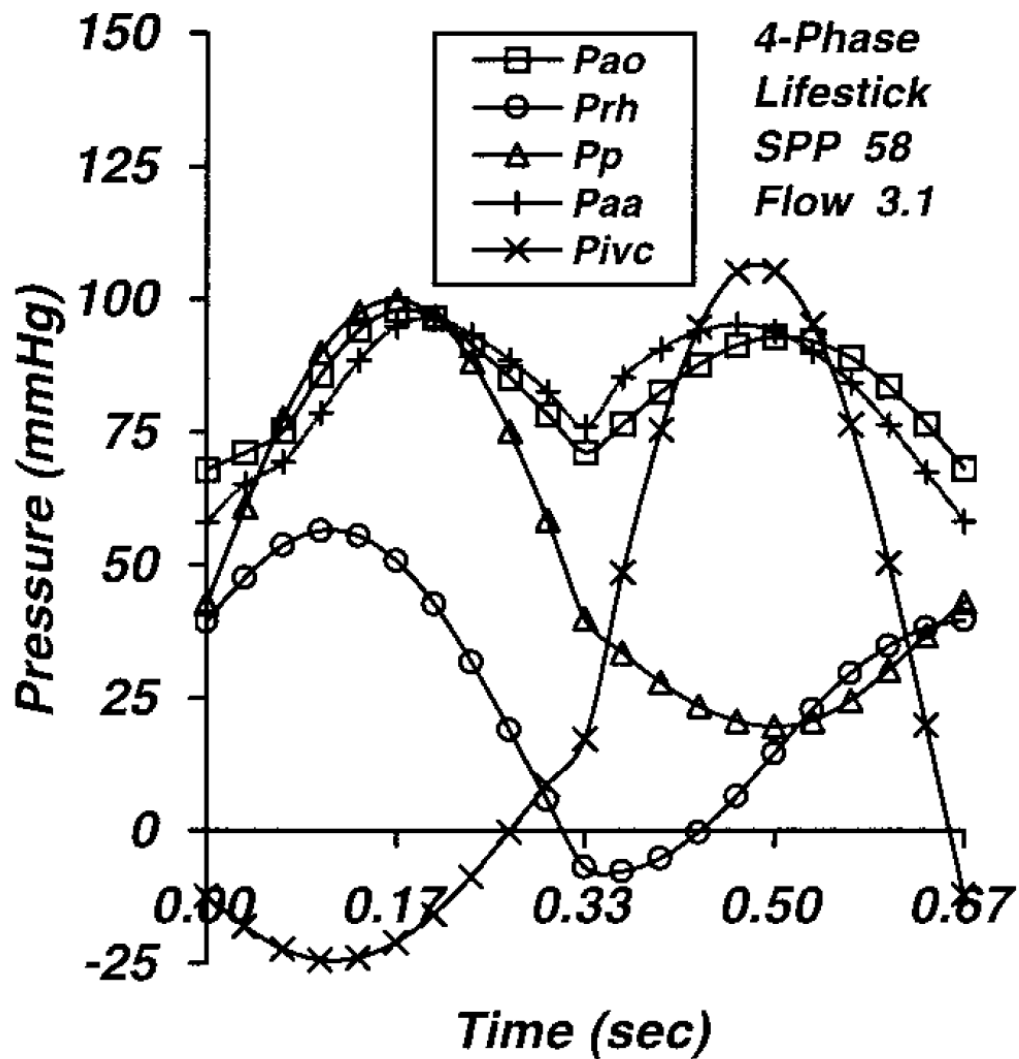


Figure 4. Pressures generated by four-phase Lifestick cardiopulmonary resuscitation in a mathematical model. Details are similar to Figure 1. P_{ao} , thoracic aortic pressure; P_{rh} , right heart pressure; P_p , thoracic pump pressure; P_{aa} , abdominal aortic pressure; P_{ivc} , inferior vena cava pressure; $Flow$, forward flow in L/min; SPP , mean systemic perfusion pressure in mm Hg.

Influence of Chest Pump Mechanisms. Systemic perfusion pressures obtained by chest and abdominal compression are dependent on the degree to which blood is impelled by cardiac compression vs. global intrathoracic pressure fluctuation. In Figure 5, mean systemic perfusion pressure is plotted as a function of the thoracic pump factor for the four possible CPR techniques: standard, IAC, ACD, and four-phase Lifestick CPR. Applied compression or decompression pressures are the same as in Figures 1–3. Although perfusion pressures for the augmented CPR techniques are always better than those for standard CPR, the ratios of experimental to standard perfusion pressures vary with the thoracic pump factor.

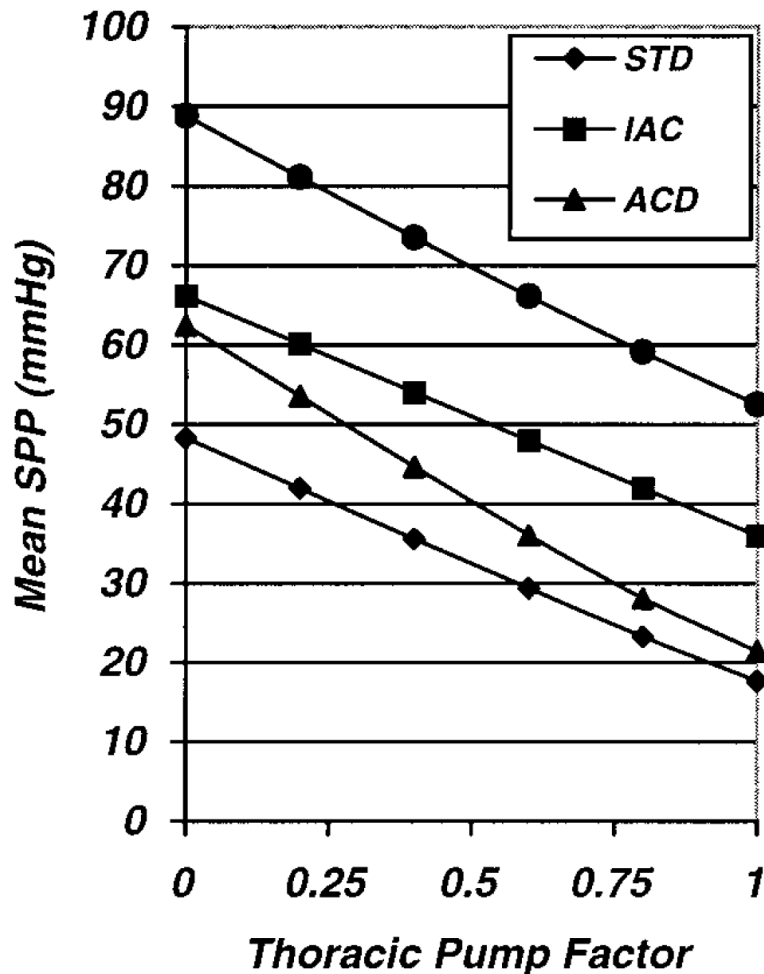


Figure 5. Mean systemic perfusion pressure (*SPP*) generated by standard and augmented cardiopulmonary resuscitation techniques in models with varying mechanisms of blood flow. Effects of chest compression vary according to the thoracic pump factor, ranging from 0 (no compression of the thoracic aorta and superior vena cava) to 1.0 (same pressure applied to the thoracic aorta and superior vena cavae as is applied to the heart). *STD*, standard; *IAC*, interposed abdominal compression; *ACD*, active compression and decompression. Filled circles indicate four-phase, Lifestick cardiopulmonary resuscitation.

The relative benefit of IAC-CPR, compared with standard CPR, evident in Figure 5, appears greater in a pure thoracic pump model than in a pure cardiac pump model. Conversely, the relative benefit of ACD-CPR appears greater in a pure cardiac pump model. The apparent benefit of ACD-CPR is especially model-dependent and may be greater in small animal models such as beagles, which permit more cardiac compression, than in larger animal models, including humans. This effect might well explain the generally more dramatic and favorable results with ACD in animal models, compared with the overall mixed results observed in humans (38).

DISCUSSION

The present mathematical analysis provides an independent test confirming the efficacy of adjunctive diastolic phase maneuvers to augment perfusion during CPR. It offers a convenient and low-cost way to compare various CPR adjuncts in exactly the same test system, eliminating the need to extrapolate published results from one animal or clinical model to another. The results confirm that compression and decompression of either the chest or the abdomen can help to move blood in cardiac arrest. Importantly, the positive effects of IAC-CPR, ACD-CPR, and four-phase Lifestick CPR are a direct consequence of normal anatomy of the circulation and two very fundamental principles of cardiovascular physiology—the definition of compliance and Ohm's law.

Analysis of pressure waveforms suggests that these techniques function primarily by pump priming. In IAC-CPR, the chest pump is primed by positive pressure in the abdomen during thoracic recoil. In ACD-CPR, the chest pump is primed by negative diastolic pressure in the chest that draws blood centrally from extrathoracic veins. In four-phase Lifestick CPR, these effects are combined so that negative thoracic and positive abdominal pressures prime the chest pump. In turn, positive thoracic and negative abdominal pressures prime the abdominal pump.

CONCLUSIONS

Improved perfusion during IAC-CPR may not necessarily lead to better long term survival, especially when the underlying rhythm is asystole or electromechanical dissociation (11). However, a nihilistic attitude toward CPR will never lead to improved resuscitation rates and is hardly justified. Systemic perfusion pressure achievable with IAC alone is approximately double that of standard CPR.

Systemic perfusion pressure achievable with full four-phase CPR might possibly exceed three-fold that of current standard CPR. Clinical data suggest that, if adjuncts like IAC-CPR are used to give improved hemodynamics, improved survival and outcome in a general population can and do result (2). The IAC, ACD, and Lifestick techniques represent the culmination of two decades of research on hemodynamic mechanisms during cardiac arrest and resuscitation. Performed by trained healthcare providers, these methods are valid and practical alternatives to standard CPR and have a rational place in resuscitation protocols of the 21st century.

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